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# AIR POLLUTION ASPECTS OF ARSENIC AND ITS COMPOUNDS

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September 1969

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# AIR POLLUTION ASPECTS

# OF

# ARSENIC AND ITS COMPOUNDS

Prepared for the
National Air Pollution Control Administration
Consumer Protection & Environmental Health Service
Department of Health, Education, and Welfare
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# FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

Aeroallergens (pollens)
Aldehydes (includes acrolein and formaldehyde)
Ammonia
Arsenic and Its Compounds
Asbestos
Barium and Its Compounds
Beryllium and Its Compounds
Biological Aerosols (microorganisms)
Boron and Its Compounds
Cadmium and Its Compounds
Chlorine Gas
Chromium and Its Compounds (includes chromic acid)
Ethylene
Hydrochloric Acid
Hydrogen Sulfide
Iron and Its Compounds
Manganese and Its Compounds
Mercury and Its Compounds
Nickel and Its Compounds
Odorous Compounds
Organic Carcinogens
Pesticides
Phosphorus and Its Compounds
Radioactive Substances
Selenium and Its Compounds
Vanadium and Its Compounds
Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are

necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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## ABSTRACT

Arsenic and its compounds are known to be toxic to humans, animals, and plants. Arsenical dusts may produce dermatitis, bronchitis, and irritation to the upper respiratory tract. Medicinal ingestion of arsenic has produced keratoses and cancer of the skin. The relationship of arsenic to other types of cancer, particularly lung tumors, is uncertain. Herbivorous animals have been poisoned after eating plants contaminated with arsenic compounds.

Arsenic is produced by smelters processing arsenical ores. Because there is no economic incentive to remove arsenic from the exhaust fumes of smelting, the smelter is a potential local pollution source.

Arsenical compounds are used as insecticides and herbicides. The use of arsenic in pesticides has declined since the appearance of organic pesticides. The largest quantity of arsenic is used as a desiccant for cotton prior to machine picking. As a result, arsenic air pollution occurs during cotton ginning and the burning of cotton trash.

Coal also contains a small amount of arsenic, and for this reason, most city air contains a small amount of arsenic given off by combustion of coal. The 1964 average daily concentration was 0.02 $\mu g/m^3$.

Particulate control of emissions from smelters and cotton gins appears to be adequate to control arsenic. However, hot exhaust gases must be cooled prior to removing arsenic as particulate because arsenic trioxide sublimes at 193°C.

No information has been found on the economic costs of arsenic air pollution or on the costs of its abatement.

Methods of analysis are available for the measurement of arsenic at concentrations in ambient air.

CONTENTS : Arsenic's and arsenic compounds!


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# 1. INTRODUCTION

Air pollution caused by arsenical compounds has been observed near gold[9,10] and copper[21,22,23] smelters as well as in the areas where arsenic is used for agricultural purposes.[40] A small amount of arsenic can be measured in the air in most cities.[2,3,55]

Arsenic is a common industrial nuisance wherever arsenical ores are smeltered.[28,29] Before the advent of organic insecticides (e.g., DDT) the use of arsenicals as pesticides was increasing. However, since then, their use has leveled off and perhaps declined as the organic pesticides have taken their place. As a result, the supply of arsenic is greater than the demand, and the only economic incentive to remove arsenic from the exhaust fumes of smelters is the presence of other trace metals, such as tellurium, selenium, tin, zinc, and antimony.

Arsenic trioxide (white arsenic, arsenious oxide, $As_2O_3$) is the common commercial form of arsenic. Most compounds of arsenic, when heated in air, are converted to this tasteless, toxic, white powder. Arsenic metal, arsenic sulfides, arsine, arsenic(V) oxides (in the presence of a reducing agent), and organic arsenates, are all converted by heat and oxygen to arsenic trioxide. Since arsenic trioxide sublimes at 193°C, it is easily suspended as small particles in the air.[28,29]

## 2. EFFECTS

The effect of arsenic on humans, animals, and plants depends on the level of concentration and particular chemical compound in which it is found. Arsine, $AsH_3$, is extremely toxic while metallic arsenic is nontoxic. While organic arsenates, such as cacodylic acid, $(CH_3)_2H\ AsO_2$, are toxic to plants, they are relatively less toxic to animals, while the reverse is true for calcium and lead arsenates. Buchanan[12] and Frost[19] have recently written excellent reviews on the toxicity and biological effects of arsenic.

### 2.1 Effect on Humans

When arsenical compounds are present in the air, arsenic may be absorbed by inhalation, ingestion, or absorption through the skin. The airborne arsenic dust frequently causes irritation of the skin and mucous membranes, absorption taking place most readily on moist surfaces such as folds in the skin or mucous membranes. Thus, dermatitis, mild bronchitis, and nasal irritation are common symptoms of arsenic poisoning. With more severe exposure, perforation of the nasal septum takes place. Other systemic symptoms (see Table 1) caused by ingestion are uncommon in people who are merely exposed to arsenic dust. Because of its irritant properties, inhalation of sufficient amounts of arsenic trioxide to cause systemic poisoning would be difficult.[4] It has been reported that the fatal dose by ingestion of arsenic trioxide for man is 70,000 to 180,000 μg.[4]

TABLE 1

SYMPTOMS OF ARSENIC POISONING[12,19]

| Acute | Chronic |
|---|---|
| Inflammation of the stomach and intestine | Weakness |
| Difficulty in swallowing | Loss of appetite |
| Burning in the throat | Nausea and occasional vomiting |
| Violent cramplike pains | Pains in stomach and intestines |
| Nausea | Diarrhea or constipation |
| Vomiting | Inflammation of mucous membrane of nose and gums |
| Diarrhea | Sore gums |
| Cold, damp skin | Runny nose |
| Feeble, irregular heartbeat | Perforation of nasal septum |
| Possible death in 1-4 days or possible chronic symptoms | Sneezing |
| | Coughing |
| | Skin ulcers |
| | Grayish pigmentation of skin |
| | Dermatitis |

The colorless gas, arsine, is responsible for a few deaths each year. Arsine is formed wherever hydrogen is produced in the presence of arsenic. Thus, in the pickling of metals containing arsenic, arsine can be formed.[5] An exposure of 3,000 to 30,000 $\mu g/m^3$ for one hour is probably dangerous, and 210,000 $\mu g/m^3$ is probably hazardous to life. Exposure to lower concentrations (less than 1,500 $\mu g/m^3$) may cause jaundice and hemolytic anemia, with the primary effect being the destruction of red blood cells.[5]

Any arsenic taken into the body is excreted primarily in the urine, but some also in the feces, hair, nails, and epithelium.[41] Arsenic may be found in small quantities in the blood, all the tissue, the bones, and especially the hair. The arsenic content of the hair has been used to determine the arsenical

exposure history and has served as evidence of homicidal poisoning. Since some arsenic is consumed in food, it would not be possible to determine the amount of atmospheric exposure from the quantity present in the hair. The biological half-life for the excretion of arsenic is 30-60 hours.

Arsenic compounds have been used medically for treatment of syphilis and skin disorders as well as to increase resistance to fatigue. Over a period of time, it appears that a tolerance can be developed to limited quantities of arsenic.[19] However, keratoses on the palms of the hands and soles of the feet often appear after prolonged use of arsenic for medicinal purposes.[12]

2.1.1 Carcinogenesis

Arsenic is one of the most controversial of known or suspected environmental carcinogens. As early as 1820, arsenical compounds were suspected of carcinogenic action.[12] This impression was based on the observation that skin cancer frequently occurred following therapeutic administration of arsenic for psoriasis and other disorders. According to Buchanan,[12] nearly all of these cases of skin cancer followed a prolonged period of medicinal administration (averaging 18 years) of inorganic trivalent arsenic. He states that cancer frequently (80 percent of published cases) follows the nonmalignant manifestation of keratosis, commonly on the palms of the hands or soles of the feet.

In 1963, Heuper[26] listed arsenic as one of the recognized human carcinogens. The skin, lung, and liver were listed as recognized sites of arsenic cancers, and the mouth, esophagus, larynx, and bladder as suspected sites.

The role of arsenic as a respiratory carcinogen has received some support from the finding of above-average mortality from lung cancer in South Rhodesian miners[38] of gold-arsenical ores and the frequent occurrence of lung cancer in German vineyard workers[11,48] exposed to lead arsenate dust.

In opposition, Frost[19] argued that the carcinogenic action was inappropriately attributed to arsenic because of the tendency to specify arsenic as the carcinogen even when other materials were present. Nickel in particular, appears to be a carcinogen which occurs together with arsenic in industrial dusts. (See the companion report on the air pollution aspects of nickel.) The strongest arguments against arsenic as a carcinogen are the failure to show increased prevalence of cancer among industrial workers and failure to induce cancer in experimental animals.

Snegireff and Lombard[52] examined the records of two industrial plants in relation to the number of employees who died of cancer. In a plant where the workers were exposed to arsenic, 18 of 146 deaths (12.3 percent) were caused by cancer. In the second plant, where the workers were not exposed to arsenic, 12 of 109 deaths (11.0 percent) were caused by cancer.

The authors concluded that there was no significant difference in cancer mortality between plant employees who handled arsenic and those who were not exposed.

In another study, Pinto and Bennett[43] compared the mortality of employees who handled arsenic for the American Smelting and Refining Company at Tacoma, Wash., with those whe were not exposed to arsenic. (This smelter is the only plant presently producing arsenic commercially in the U.S. See Section 3.) They observed that 6 of 38 deaths (15.8 percent) among workers who were exposed to arsenic trioxide were caused by cancer, while 37 of 191 deaths (19.4 percent) were due to cancer among workers not exposed to arsenic. The evidence that these arsenic workers were exposed to higher concentrations of arsenic was confirmed by urinalysis. This lower percentage of deaths among arsenic workers compared favorably with the male deaths due to cancer (15.9 percent) in the entire state of Washington.[43] Arsenic workers excreted an average of 820 µg/liter of arsenic in the urine compared to 130 µg/liter for unexposed workers. The authors found no evidence that arsenic trioxide caused systemic cancer or fatal cardiovascular disease in humans.

Attempts to demonstrate through animal studies that arsenic is carcinogenic have often met with failure.[7,12,27,29,50] In fact, one study showed that arsenic suppressed the appearance of spontaneous tumors of the lung.[27] However, a few cases have been reported in which arsenical cancer was induced in animals.[12]

(See Section 2.2.2)

Some investigators have mentioned that the type of arsenic compounds involved may play a role in the carcinogenesis. Cornelius and Shelley[17] suggest that arsenic trioxide to which most smelter workers are exposed is probably not as carcinogenic as other soluble arsenic compounds. The composition and effect of arsenic compounds found in the ambient air have not been determined.

This dispute regarding the relationship between arsenic and cancer is probably the most important question in relation to the air pollution aspects of arsenic.

2.1.2 Community Episodes

In June 1962, a gold mine and smelter[9,10] in one of the Western States were reopened; after they were in operation for approximately 5 months it became apparent that the emission control equipment was inadequate. Both sulfur dioxide and arsenic trioxide were emitted in visible quantities into the atmosphere. Air samples taken at the plant showed 60 to 13,000 $\mu g/m^3$ of arsenic. A yellowish-gray dust on the ground gave evidence of fallout from the plant. No air samples were taken in the small mining community adjacent to the plant. However, the results of analyses of dust, water, and grass samples in the area are shown in Table 2.

A clinical examination was made of about 40 children attending two schools. Thirty-two of the children had

TABLE 2

ARSENIC CONTAMINATION IN A WESTERN MINING COMMUNITY[9,10]

| Sample | Site | Arsenic Concentration |
|---|---|---|
| Flue dust | Base of stack | 44% |
| Roof dust | Shed near office | 4.4% |
| Dust | Area near drying mill | 2.7% |
| Dust | Roaster area | 3.1% |
| Dust | Sulfide-ore feed | 1.23% |
| June grass | Area near school | 925 µg/g |
| Water | Tap water | 30 µg/l |

dermatosis associated with cutaneous exposure to arsenic. At first, it was thought that this was an example of systemic arsenic poisoning; however, it proved to be a contact dermatitis. Local skin irritation was observed in the folds of the skin and where the skin was moist. In a few cases the conjunctivae and nasal mucosae were irritated. It is interesting to note that there were no cases of dermatitis among the older children who were bussed to a high school in a distant town: the disease occurred only among the younger children who spent all their time in town. Moreover, no new cases occurred after controls were introduced at the plant. Keratoses, epitheliomas, and melanodermas were not present. There were, of course, typical symptoms resulting from exposure to arsenic, such as skin disease and nasal septum perforation, among the refinery workers. Although animals were not systematically studied, the pet population of more than two dozen had been reduced to one dog.

Another example of arsenical air pollution occurred in Montana[21,22,23,57] in the years 1903 to 1905. During this time, large quantities of arsenic (see section 3.2) were emitted from a copper smelter. Although large numbers of animals were killed from eating plants contaminated with arsenic trioxide, no record of human health complaints is available.

An arsenical air pollution episode occurred at a copper mine in northern Chile.[39] The concentrations of arsenic present during this incident are shown in Table 3. A survey of 124 workers showed arsenical melanosis in 7.25 percent, arsenical dermatitis in 5.65 percent, and perforation of the nasal septum in 1.6 percent. No cutaneous manifestations were encountered among a control group or among members of the mining community.

TABLE 3

THE CONCENTRATION OF ARSENIC NEAR A COPPER MINE IN NORTHERN CHILE[39]

| Sample | Arsenic Concentration |
|---|---|
| Mineral (ore) | 0.054% |
| Concentrated ore | 1.64% |
| Calcined ore | 0.30% |
| Dust from electrostatic precipitator | 10.36% |
| Dust from stack | 16.64% |
| Soil in plant | 1,000 μg/g |
| Soil on road to plant | 650 μg/g |
| Soil near hospital | 20 μg/g |
| Soil near workers' club | 90 μg/g |
| Air at roasting plant | 400-81,000 $\mu g/m^3$ |
| Air at smelter plant | 400-5,400 $\mu g/m^3$ |

## 2.2 Effects on Animals

### 2.2.1 Commercial and Domestic Animals

In the preceding section, the three community episodes which were cited indicate that arsenical air pollution may have deleterious effects on animals. In the Chilean[39] episode, it was noted that dogs and chickens suffered from ulcers of the feet, although it was not definite that these lesions were due to contact with arsenic. In the Western State gold-mine episode,[9,10] the pet population was reduced from over 24 to 1. The surviving collie had a large ulcer in the mouth and another on the right forepaw.

The Montana[21,22,23,57] episode caused widespread damage to herbivorous animals caused by the ingestion of arsenic trioxide, which had contaminated the forage crops. Cows, sheep, and horses suffered from symptoms similar to those often observed in humans exposed to arsenic. When a flock of 3,500 sheep was brought from an area 28 miles away to graze 15 miles from the smelter, 625 of the sheep died. Upon analysis, grass and moss from the area in which the animals had fed was found to contain 52 and 405 ppm of arsenic trioxide respectively. Moreover, horses in an area remote from the smelter died after eating hay grown in a location on which the smelter fumes could have fallen. The arsenic trioxide content of the hay was 285 ppm. In addition, large quantities of both arsenic (10-150 ppm) and copper (128-1,800 ppm) were found on the vegetation up to 15 miles from the smelter.

Both the type of symptom and data from analyses of tissues failed to implicate copper as the poisoning agent, whereas evidence concerning animals fed on graded doses of arsenic verified experimentally that arsenic was responsible.

A study of the effects of airborne arsenic from a nearby smelter on animals in Saxony Forest was reported by Prell.[44] Red deer, foxes, and horses were all affected. The deer showed signs of thickened skins and joints, malformation of the horns, and loss of hair. The arsenic content of the stomach, intestine, liver, kidneys, and hair of the different species varied from traces to 42,000 μg/kg of tissue. Bees in the area had a high mortality rate. Analysis showed as much as 1 μg of arsenic per bee and 88 μg of arsenic per g of pollen.

Arsenical compounds, especially lead and calcium arsenate, have been used as insecticides. Most arsenates are toxic to insects and have little effect on plants. If ingested in sufficiently large quantities, the other compounds of arsenic are fatal to insects.

Aquatic animals have a higher tolerance for arsenic and normally have higher arsenic contents in the tissue than other animals.[12] For this reason, arsenicals have been recommended for use in the control of aquatic weeds.[50]

### 2.2.2 Experimental Animals

Frost[19] has reviewed the literature on the carcinogenic effects of arsenic. He reports that more than 35 experiments

yielded negative results for carcinogenicity when mice, rats, pigs, and dogs were tested with arsenic trioxide, potassium arsenate, sodium arsenite, sodium arsenate, arsanilic acid, 3-nitro-4-hydroxyphenylarsonic acid, and p-dimethylaminoazobenzene-p[1]-arsononic acid.

Buchanan[12] points out that Leitch and Kennaway[31] succeeded in inducing only one squamous epithelioma (after 86 applications of potassium arsenite) in 100 mice. Several other experimenters are also cited by Buchanan as follows. Raposo[46] developed hyperplasias and 3 papillomas in the ears of 10 rabbits painted with arsenious oxide. Cholewa[14] painted the ears of two rabbits with potassium arsenite and observed a papillomatous wart and a sarcoma of the perichondrium in the ears of one rabbit after one year. Askanazy[6], Goeckerman, and Wilhelm[20] observed teratogenic effects when rats receiving transplanted embryos drank water containing arsenic. Holmberg et al.[25] injected pregnant hamsters with barely sublethal doses (20,000 µg/kg) of sodium arsenate. Of 177 embryos, 49 percent were malformed and 84 percent were either malformed or resorbed. The abnormalities observed were encephalocele, exencephaly, unilateral and bilateral eye and ear malformations, and cleft palate or lip. Exencephalic malformations were particularly prominent in all litters. When sodium selenite (2,000 µg/kg) was injected simultaneously with sodium arsenate (20,000 µg/kg) into 12 pregnant hamsters, only 19 percent of 144 embryos showed malformations and 39 percent, malformation or resorption.

Thus, these data confirm the work of Ferm and Carpenter[18] that arsenic is teratogenic as well as the fact that selenium is antagonistic to arsenic.

Recently Milner[34] experimented with three strains of mice, CXC3H, DBA, and Balb/C. Tumors were induced with methylcholanthrene or promoted by grafting the methylcholanthrene-treated skin on the flanks of recipient animals. Approximately 20 animals were tested in each experiment. Animals tested were given arsenic trioxide in their drinking water. The only significant effect observed by Milner was that the CXC3H strain of mice showed a reduction in papillomas. Thus, arsenic appeared to have little effect on the development of tumors in mice.

The above results tend to support the work of Kanisawa and Schroeder[27] who observed that mice fed 0.46 μg/gm sodium arsenite developed fewer spontaneous tumors (11/103) than the controls (55/170). However, only the number of adenomas and carcinomas of the lung was significantly lower.

## 2.3 Effects on Plants

In the smelter episodes discussed in Section 2.1.2 plant damage has been observed. However, according to Birmingham et al.[10] sulfur dioxide present in the air, rather than the arsenic, is more likely to have caused the damage.

Sodium arsenite is used as a soil sterilant to control vegetation around fence posts, bridge abutments, radar sites, tennis courts, roadways, and other nonagricultural areas.[50]

Sprays used to control weeds have reportedly damaged some crops in adjacent farms.

Organic arsenicals have been synthesized and are used to defoliate cotton plants prior to machine picking, to kill potato vines prior to machine picking, to control aquatic weeds, and to a limited extent to defoliate other plants.[50] Thus, arsenical compounds can be harmful to plants if their use is not properly controlled.

## 2.4 Effects on Materials

No information has been found on the effects of arsenic on materials. However, arsenicals are used as preservatives. For instance, arsenates are used as wood preservatives,[50] especially against termites. Arsenical paints have been used in the past, but they have been replaced by other materials.

## 2.5 Environmental Air Standards

No 24-hour maximum atmospheric concentration has been set in the United States for arsenic. A basic 24-hour standard of 3 $\mu g/m^3$ for arsenic and its compounds (as arsenic) has been recommended in the U.S.S.R.[47,49] and Czechoslovakia.

The threshold limit values recommended for industrial workers (8-hour/day exposures) by the American Conference of Governmental Hygienists[28] are 500 $\mu g/m^3$ for arsenic and its compounds (as arsenic) and 200 $\mu g/m^3$ for arsine. The American Industrial Hygiene Association recommends the same values.[5]

Emission standards have been summarized by Stern.[54] These values are listed in Table 4.

TABLE 4

EMISSION STANDARDS FOR ARSENIC IN EFFLUENT AIR OR GASES[54]

| Location | Source of Emission | Standard Original Units | Standard µg/m³ |
|---|---|---|---|
| Czechoslovakia | | 0.03 kg/hr | |
| Great Britain | < 5,000 cfm | 0.05 grains/ft³ | 115,000 |
| Great Britain | > 5,000 cfm | 0.02 grains/ft³ | 46,000 |
| New South Wales | | 0.01 grains/ft³ | 23,000 |
| Queensland | | 0.01 grains/ft³ | 23,000 |

## 3. SOURCES

There are three major sources of arsenic air pollution: smelting of metals, burning of coal, and use of arsenicals as pesticides.

### 3.1 Natural Occurrence

Arsenic is so widely distributed that traces of it can be found almost everywhere. However, in terms of its concentration in the earth's crust (approximately 5 μg/g), it is one of the less plentiful elements. Virgin soils usually contain a few ppm of arsenic.[28]

Arsenic is present in sea water (10 to 100 ppb) and is concentrated in some aquatic creatures, such as shrimp. These supply man with an appreciable percentage of his total intake of arsenic.[28]

Arsenic is commonly found as the sulfide, arsenide, arsenite, or arsenate. Occasional deposits of elemental arsenic are found, but none are commercially important.[29]

### 3.2 Production Sources

Virtually all of the arsenic produced is recovered as a by-product in the smelting of lead, copper, and gold ores. The production of white arsenic as a by-product has been so great that the supply usually exceeds the demand.[30] Until this year, the United States' domestic needs have been supplied by the Anaconda Company at Anaconda, Mont. and the American Smelting and Refining Company at Tacoma, Wash.,

supplemented by some imports. However, the Bureau of Mines reports that the Anaconda Company suspended its sale of arsenic in 1968.[15]

In order to avoid disclosing company confidential data, the U.S. consumption or production of white arsenic has not been reported since 1959. Prior to that, the U.S. consumption varied between 13,000 and 40,000 short tons per year.[35]

The price of arsenic has declined from approximately 6.5 cents per pound to about 4 cents.[15,30]

One of the problems facing these mining industries has been the disposal of the large quantities of arsenic they produce.[28,29] A gold smelter in a small Western town produced 14,600 tons per year, almost enough to supply all our domestic needs. These industries are also faced with the disposal of the very poisonous arsenic trioxide.[9,10]

The high volatility of arsenic trioxide (sublimes at 193°C) requires that most arsenic-containing ores be specially treated to remove arsenic from the exhaust gases. Lead, copper, and gold ores may contain up to 3 percent arsenic.[28,29] Arsenic is also a contaminant in some nickel and cadmium ores, and must be removed to improve the quality of the metal. In some processes the arsenic is removed chemically, while in others it is removed by taking advantage of the high volatility of the arsenic trioxide. Since the latter process provides a most important source of air pollution, it will be discussed in detail.

In the commercial production of arsenic, arsenic trioxide is volatilized during the smelting process and concentrated in the flue gases. Crude flue gas dust may contain up to 30 percent arsenic trioxide, the balance being oxides of copper or lead and perhaps of other metals, such as antimony, tin, and zinc. To upgrade the flue dust, a small amount of pyrite or galena is mixed with the concentrate and the mixture roasted. The gases are finally passed through a series of brick cooling chambers called kitchens. The temperatures of the gas and vapor are controlled; they enter the first kitchen at approximately 220°C, and by the time the gas and vapor reach the last kitchen, they have been cooled to 100°C or less. The condensed crude product is 90 to 95 percent arsenic trioxide. Resublimation at about 295°C and recondensing in kitchens at 180 to 120°C produce 99 to 99.9 percent arsenic trioxide.

Even in the smelters where arsenic is not recovered for commercial use, the tonnages involved are very large. A reverberatory furnace, for example, may smelt as much as 2,100 tons of charge per day, and in doing so, burn 240 tons of coal. The furnace would produce about 90,000,000 cubic feet of gas per day, containing 180 tons of solids. This means that it would be necessary to dispose of up to 60 tons of arsenic daily.[28,29]

Two serious arsenic air pollution incidents from smelters in the United States have been recorded in the literature, both of which were mentioned in the preceding

section. The first incident took place in Anaconda, Mont.,[21,22,23] where the emission rate of arsenic trioxide was 59,270 pounds per day (in 2.28 X $10^6$ $ft^3$ of air per day) while processing 10,000 tons of copper ore per day. This resulted in polluting the air at the exit of the stack with approximately 450 $g/m^3$ of arsenic trioxide (estimated by author); the air was then dispersed over a radius of 15 miles. Although no atmospheric concentrations are recorded, the edible plants were contaminated by as much as 482 µg of arsenic trioxide per gram of plant. It is noteworthy to reiterate that while the animals eating these plants were killed at distances up to 15 miles from the smelter, no human health complaints are recorded.

The second incident, previously mentioned, occurred in a small Western town near a gold smelter.[9,10] (The exact location is not mentioned.) The mine had been intermittently operated since 1934. In 1962, the operation was resumed with a process that required converting the sulfur and arsenic to sulfur dioxide and arsenic trioxide to successfully accomplish subsequent cyanidation of the gold. The smelter processed sufficient ore to produce about 100 tons of sulfur dioxide and 40 tons of arsenic trioxide per day. The dust-collecting system which was intended to collect approximately 90 percent of the toxic dusts failed to operate as expected and toxic fumes escaped into the atmosphere.

These two episodes indicate that there is an arsenical air pollution potential at every smelter which refines arsenical

ores. An example of the arsenical pollution estimated for Colorado[29] in 1963 is given in Table 5.

TABLE 5

ARSENICAL POLLUTION POTENTIAL FROM MILLS IN COLORADO, 1963[29]

| Metal | Average Arsenic Content (Percent) | Ore (short tons) | No. of Mines | Potential Arsenic Pollution (tons) |
|---|---|---|---|---|
| Zinc | 0.07 | 48,109 | 8 | 34 |
| Lead | 0.08 | 19,918 | 8 | 16 |
| Copper | 0.28 | 4,169 | 1 | 12 |
| Total | | | | 62 |

Arsine is produced whenever hydrogen is emitted from the dissolution of arsenical metals, such as in metal pickling, soldering, etching, or in plating processes involving metals or acids containing arsenic. This constitutes an industrial hazard but is not an air pollution problem since the quantities are usually very small.[28,29] Some years ago, arsine was found to be produced by molds growing on wallpaper which had been colored with arsenical pigments. Since this procedure is no longer in use, a hazard no longer exists. However, based on this evidence an arsenic cycle has been hypothesized in which arsine is emitted into the atmosphere, oxidized in the presence of light to form arsenic trioxide deposits on plants, eaten by animals, and eventually returned to the earth, where it can be reduced to arsine again.[19]

## 3.3 Product Sources

### 3.3.1 Pesticides

Arsenical pesticides constitute the primary use of arsenic. Until 1945, when DDT made its appearance, the use of arsenical insecticides was increasing. DDT or other organic insecticides have almost replaced arsenical insecticides. Arsenical herbicides were also replaced by organic herbicides such as 2,4-D acid, which appeared on the market about the same time as DDT. The production of arsenical pesticides is summarized in Table 6 in the Appendix.

Several arsenical compounds are currently recommended by the U.S. Department of Agriculture for insecticides and herbicides. The compounds listed in Table 7 (Appendix) are recommended.[56] In 1964, arsenic acid was the largest volume product in the defoliant-desiccant category. About 5.0 million pounds were used on about 1.2 million acres of cotton.[45] Table 8 in the Appendix lists the quantities of arsenical pesticides used by farmers.[45]

In 1968 the military used cacodylic acid extensively to control vegetation around encampments. Another domestic demand for cacodylic acid was for weed control around industrial sites, rights-of-way, and fence rows. Forest insect, forest disease, and timber management also used cacodylic acid, in the amounts of 815 pounds in 1967 and 173 pounds in 1968.[42]

From 1937 to 1940 the U.S. Public Health Service[36] studied the effect of lead arsenate insecticides on orchardists. During this study, they measured the concentration of arsenic and lead to which the orchardists were exposed. These data are summarized in Table 9 in the Appendix. No data were given indicating the pollution area or concentration of arsenic downwind from the various operations. While the authors do not emphasize the air pollution caused by burning, it is noteworthy that the highest concentrations of arsenic were measured in the smoke from burning the pesticide containers. Moreover, the arsenic-to-lead ratios are out of proportion to the other concentrations. The high arsenic concentrations might be explained by the volatility of arsenic trioxide which is formed in the burning process.

### 3.3.2 Cotton Gins

Arsenicals are used for weed control and as desiccants for cotton plants prior to machine picking.[16,40,50] Thus, the dust emitted from cotton gins contains arsenic. Table 10 in the Appendix shows the concentration of dust and arsenic observed near a cotton gin in Texas and indicates that the arsenic content is approximately 0.03 percent of the particulate. Table 11 (Appendix) shows that the particulate emission concentration from the Stoneville gin may range between 11,000 and 1,258,000 $\mu g/m^3$. On the assumption that the concentration of arsenic is approximately 0.03 percent by

weight of the particulate, one can estimate that the Stoneville plant would emit a maximum of 400 $\mu g/m^3$ of arsenic in the air exhausted from the gin and a minimum of 3 $\mu g/m^3$. The range of air volume exhausted from a gin is 1,410 to 2,120 per cubic meter per minute or 9,150 to 15,900 per cubic meter per bale of cotton. This would result in emission rates of up to 580,000 µg/min or 6,360,000 µg/bale as shown in Table 12 in the Appendix.

In addition to the operation of the cotton gin, the burning of trash from a cotton gin is also a source of arsenic pollution.[53] The fieldmen of the Cotton-Classing offices[16] have indicated that approximately 37 percent of the gins incinerate the trash, 58 percent return it to the land, and 5 percent handle it in some other manner. No estimate was made of the arsenic emissions from incineration. However, one might expect that all of the arsenic present in the burning trash would be converted to volatile arsenic trioxide, which is then emitted into the atmosphere. Arsenic has been observed in the smoke from burning cotton burr trash.[53] Adverse effects on trees and vegetation in areas downwind from cotton gins were observed. Peach trees were killed by arsenic and pecan trees damaged. These observations were confirmed by laboratory analyses. The control of cotton gin dust is not enough; the emissions from burning trash must also be controlled.

### 3.4 Other Sources

Coal contains 0.08 to 16 µg of arsenic per gram of coal.[1] Therefore, the air of most cities contains a small

amount of arsenic. Analyses of the dusts in Hamburg, Germany[1] and Leeds, England,[1] have shown that dusts contained 30 to 230 μg of arsenic per gram. With approximately 409 million tons[51] of coal used each year in the United States, it is possible for 327 to 6,440 tons of arsenic to be emitted into the atmosphere each year. New York City used approximately 5.8 million tons[37] of coal in 1966. This could have resulted in 4.6 to 93 tons of arsenic per year being emitted into New York City air. The average particulate concentration in New York City[37] in 1966 was approximately 125 $\mu g/m^3$. Assuming that the European values for arsenic in dust hold for New York City, values of 0.004 to 0.029 $\mu g/m^3$ of air can be calculated. This is in agreement with the value of 0.03 $\mu g/m^3$ reported in Tables 11 and 12 (Appendix) for New York City.

3.5 Environmental Air Concentrations

Air quality data taken in 1950, 1953, 1961, and 1964 of the arsenic concentration are shown in Tables 13, 14, 15, and 16 (Appendix) respectively. Of the 133 stations reporting in 1964 the year average ranged from below detection to 0.75 $\mu g/m^3$ and an average for all stations of approximately 0.02 $\mu g/m^3$. The highest value given in 1964 was 1.40 $\mu g/m^3$ for quarterly average in El Paso, Texas. The Montana State Board of Health[55] also reported ambient air concentrations for certain cities in Montana for 1961-62 (see Table 17, Appendix). The highest concentration was 2.5 $\mu g/m^3$ in the city of Anaconda.

## 4. ABATEMENT

In general, the removal of particulate material will control arsenic emissions if the control equipment operates at a temperature low enough (~100°C) to condense the arsenic fumes. An electrostatic precipitator has been reported to reduce the arsenic from 5-17 ppb to 0-4 ppb.[13] Cooling flues, bag houses, and electrostatic precipitators have been used in the smelting industry.[19,29] No data have been reported in the United States on their removal efficiency for arsenic. However, at a chemical plant in the U.S.S.R., the efficiency for arsenic removal was greatly improved by using wet vacuum pumps instead of fabric filters. When the fabric filters were used, the arsenic content in the air frequently reached several hundred thousand micrograms per cubic meter. After the wet scrubbing vacuum pumps were installed, the removal is reportedly 100 percent effective.[32]

In the cotton industry, removal of particulate material emitted from cotton gins should control the arsenic emissions.[16] However, methods need to be developed to control the arsenical emissions produced by the burning of cotton trash.[50]

## 5. ECONOMICS

No information has been found on the economic costs of arsenic air pollution or on the costs of its abatement.

The production and consumption data for arsenic have been discussed in Section 3.

# 6. METHODS OF ANALYSIS

## 6.1 Sampling Methods

Dusts and fumes of arsenic compounds may be collected by any method suitable for collection of other dusts and fumes; the impinger, electrostatic precipitator, and filters are commonly used. The National Air Sampling Network uses a high-volume filtration sampler.[58]

## 6.2 Quantitative Methods

Several methods are available for detecting trace amounts of arsenic in dusts; however, only a few of these are quantitative. The chemical methods generally rely on the reduction of arsenical compounds to arsine. The arsine is transported as a gas from the reaction vessel to a second reaction chamber, where it reacts with copper foil (Reinsch's method) or is heated to produce metallic arsenic (Marsh's test), silver nitrate or mercuric chloride (Gutzeit's test), and silver diethyldithiocarbamate (ACGIH tests).[4,12] The National Air Pollution Control Administration uses silver diethyldithiocarbamate in the second reaction vessel. Neutron activation methods are both quantitative and extremely sensitive, but they require a neutron source. They are sensitive to approximately 0.1 μg of arsenic, corresponding to 0.24 $\mu g/m^3$ in a 30 cubic foot air sample.[12]

Thompson *et al.*[58] have reported that the National Air Pollution Control Administration uses atomic absorption to

supplement analyses obtained by the Gutzeit method. The method has a minimum detectable limit of 0.02 $\mu g/m^3$ based on a 2,000 cubic meter air sample.

## 7. SUMMARY AND CONCLUSIONS

Arsenic is toxic to some degree in most chemical forms. Arsenical compounds may be ingested, inhaled, or absorbed through the skin. Industrial exposure to arsenic has shown that it can produce dermatitis, mild bronchitis, and other upper respiratory tract irritations including perforation of the nasal septum. However, because of the irritant qualities of arsenic, it is doubtful that one could inhale sufficient amounts to produce systemic poisoning.

Skin cancer can result from prolonged therapeutic administration of arsenic. Similar cancers have not been observed among industrial workers. Moreover, lung tumors which resulted from inhaling mixed industrial dusts were often thought to be the result of inhaling arsenic. Recently, this relationship has been questioned because animal experiments have failed to demonstrate that arsenic is a carcinogen. Therefore, the causal relationship between cancer and arsenic is disputed.

Arsenic is poisonous to both animals and plants, but no damage to materials was found.

Two air pollution episodes in the United States have shown that there is an arsenical air pollution potential at every smelter which refines arsenical ores.

Arsenical compounds are used as insecticides and herbi-

cides. Although the use of arsenical pesticides declined sharply after the appearance of DDT and 2,4-D, arsenical compounds are still used as desiccants, herbicides, and sterilants. Some undetermined amounts of air pollution take place during spraying and dusting operations with arsenical pesticides. Pollution from cotton gins and cotton trash burning has been cited as an important source of agricultural pollution. While the emission rates from cotton trash burning have not been determined, as much as 1,258,000 $\mu g/m^3$ of exhaust air (580,000 $\mu g/min$) may be emitted during the ginning operation. This produced concentrations of only 0.14 $\mu g/m^3$ of arsenic in the air 150 feet from the gin.

Arsenic is found to the extent of approximately 5 $\mu g/g$ in coal. Therefore, the air of cities which burn coal contains some arsenic. Air quality data from 133 sites monitored by the National Air Sampling Network showed an average daily arsenic concentration of 0.02 $\mu g/m^3$ in 1964.

Control of arsenic emissions requires special attention to the temperature of exhaust gases since arsenic trioxide sublimes at 192°C. For this reason exhaust fumes must be cooled to approximately 100°C prior to removing them as particulates.

No information has been found on the economic costs of

arsenic pollution or on the costs of its abatement.

Analytical methods are available to determine arsenic at the concentration found in ambient air.

Based on the material presented in this report, further studies are suggested in the following areas:

(1) Determination of the carcinogenic effect of long-term exposure to low concentrations of arsenic in the atmosphere.

(2) Measurement of the concentration of arsenic near smelters, pesticide dusting and spraying operations, cotton gins, and places where cotton trash is burned.

REFERENCES


1. Abernethy, R. F., and F. H. Gibson, Rare Elements in Coal, U.S. Bur. Mines Inform. Circ. 8163 (1963).

2. Air Pollution Measurements of the National Air Sampling Network--Analyses of Suspended Particulates 1963, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Cincinnati, Ohio (1965).

3. Air Quality Data, 1964-1965, National Air Sampling Network, U.S. Dept of Health, Education, and Welfare, Public Health Service, Cincinnati, Ohio.

4. Arsenic and Its Compounds, Hygienic Guide Series, Am. Ind. Hyg. Assoc. J. 25:610 (1964).

5. Arsine, Hygienic Guide Series, Am. Ind. Hyg. Assoc. J. 26: (4)438 (1965).

6. Askanazy, M., Das Esperimentelle Karzinom, Schweiz Med Wochschr. 57:1209 (1927).

7. Baroni, C., G. J. Van Esch, and U. Saffiotti, Carcinogenesis Tests of Two Inorganic Arsenicals, Arch. Environ. Health 7: 668 (1963).

8. Barrows, H. L., The Agricultural Significance of Inorganic Pollutants, Presented at the Symposium on Damage to Plants from Soil and Water Pollution, American Phytopathological Society, Washington, D.C. (1967).

9. Birmingham, D. J., M. M. Key, and D. A. Holaday, An Outbreak of Dermatoses in a Mining Community--Report of Environmental and Medical Surveys, U.S. Dept. of Health, Education, and Welfare, TR-11 (April 1954).

10. Birmingham, D. J., M. M. Key, D. A. Holaday, and V. B. Perone, An Outbreak of Arsenical Dermatosis in a Mining Community, Arch. Dermatol. 91:457 (1965).

11. Braun, W., Carcinoma of Skin and Internal Organs Caused by Arsenic, German M. Monthly 3:321 (1958).

12. Buchanan, W. D., Toxicity of Arsenic Compounds (New Jersey: Van Nostrand, 1962).

13. Chambers, L. A., M. J. Foter, and J. Cholak, A Comparison of Particulate Loadings in the Atmospheres of Certain American Cities, Proc. Natl. Air Pollution Symp., 3rd, Pasadena, Calif. pp. 24-32 (1955).

14. Cholewa, J., Experimentelle Blastome allein durch chronische Arsenvergiftung, Z. Krebsforsch 4:497 (1935).

15. Cole, J. W., Bureau of Mines, Washington, D.C., personal communication (Nov. 1968).

16. Control and Disposal of Cotton-ginning Wastes, A Symposium, Public Health Serv. Publ. 999-AP-31 (May 3-4, 1966).

17. Cornelius, C. E., and W. B. Shelley, Syndactylism, Dyschromia, and the Arsenical Dilemma, Arch. Dermatol. 98:107 (1968).

18. Ferm, V. H., and S. J. Carpenter, Malformation Induced by Sodium Arsenate, J. Reprod. Fertility 17:199 (1968).

19. Frost, D. V., Arsenical in Biology—Retrospect and Prospect, Federation Proc. 26:194 (1967).

20. Goeckerman, L. H., and L. F. X. Wilhelm, Arsenic as the Cause of Cancer of Mucous Membrane: Report of a Case, Arch. Dermatol. Symphilol. 42:261 (1940).

21. Harkins, W. D., and R. E. Swain, The Determination of Arsenic and Other Solids on Smelter Smoke, J. Am. Chem. Soc. 29:970 (1907).

22. Harkins, W. D., and R. E. Swain, The Chronic Arsenical Poisoning of Herbivorous Animals, J. Am. Chem. Soc. 30:928 (1908).

23. Haywood, J. K., Injury to Vegetation and Animal Life by Smelter Fumes, J. Am. Chem. Soc. 29:998 (1907).

24. Headley, J. C., and J. N. Lewis, The Pesticide Problem: An Economic Approach to Public Policy (Baltimore: John Hopkins Press, 1967).

25. Holmberg, R. E., and V. H. Ferm, Interrelationships of Selenium, Cadmium, and Arsenic in Mammalian Teratogenesis, Arch. Environ. Health 18:873 (1969).

26. Hueper, W. C., Environmental Carcinogenesis in Man and Animals, Ann. N.Y. Acad. Sci. 108:963 (1963).

27. Kanisawa, M., and H. A. Schroeder, Life Term Studies on the Effects of Arsenic, Germanium, Tin and Vanadium, Cancer Res. 27:1192 (1967).

28. Kirk, R. E., and D. F. Othmer, Encyclopedia of Chemical Technology (New York: Interscience Encyclopedia, 1948).

29. Kirk, R. E., and D. F. Othmer, Encyclopedia of Chemical Technology, 2nd ed. (New York: Interscience Encyclopedia, 1962).

30. Lausche, A. M., "Arsenic," in Mineral Facts and Problems, Bureau of Mines Bulletin 630, U.S. Govt. Printing Office, Washington, D.C. (1965).

31. Leitch, A., and E. L. Kennaway, Experimental Production of Cancer by Arsenic, Brit. Med. J. ii:1107 (1922).

32. Matsak, V. G., The Utilization of Air Dust and Smoke Purification Equipment, Survey of the U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3:141 (1960).

33. The Merck Index, 8th ed. (Rahway, N. J.: Merck, 1968).

34. Milner, J. E., The Effect of Ingested Arsenic on Methylcholathrene — Induced Skin Tumors in Mice, Arch. Environ. Health 18:7 (1969).

35. Minerals Yearbook, Bureau of Mines, U.S. Govt. Printing Office, Washington, D.C. (1948-1966).

36. Neal, P. A., et al., A Study of the Effect of Lead Arsenate Exposure on Orchardists and Consumers of Sprayed Fruit, U.S. Public Health Service, Public Health Bull. 267 (1941).

37. New York-New Jersey, Air Pollution Abatement Activity, Particulate Matter, Phase II, Pre-conference Investigations, U.S. Dept. of Health, Education, and Welfare, Public Health Service, National Center for Air Pollution Control (Dec. 1967).

38. Osburn, H. S., Cancer of Lung in Gwanda, Central African J. Med. 3:215 (1957).

39. Oyanguren, H., and E. Perez, Poisoning of Industrial Origin in a Community, Arch. Environ. Health 13:185 (1966).

40. Paganini, O., Air Pollution Study of Cotton Gins in Texas (Progress Report), Texas State Department of Health, Austin, Division of Occupational Health and Radiation Control (1967).

41. Patty, F. A., Industrial Hygiene and Toxicology, vol. II, 2nd ed. (New York: Interscience, 1963).

42. The Pesticide Review-1968, U.S. Department of Agriculture, Stabilization and Conservation Service, Washington, D.C. (1968).

43. Pinto, S. S., and B. M. Bennett, Effect of Arsenic Trioxide Exposure on Mortality, Arch. Environ. Health 7:583 (1963).

44. Prell, H., Die Schadiging der Tierwelt durch die Pernwirkengen von Industrieabgasen, Arch. Gewerbepathol. Gewerbehyg. 7:656 (1937).

45. Quantities of Pesticides Used by Farmers in 1964, Agriculture Economic Report No. 131, Economic Research Service, U.S. Dept. of Agriculture (1968).

46. Raposo, L. S., Le Cancer a 1' Arsenic, Compt. Rend et mim Soc. Biol. 98:86 (1928).

47. Rossano, A. T., Jr., Analysis and Comparison of Available Data on Air Quality Criteria in Member Countries, Preprint, Dept. of Civil Engineering, Washington University, Seattle, Wash. (1963).

48. Roth, F., Sequelae of Chronic Arsenic Poisoning in Moselle Vintners, German M. Monthly 2:172 (1957).

49. Ryazanov, V. A., Sensory Physiology as Basis for Air Quality Standards, Arch. Environ. Health 5:86 (1962).

50. Shaw, W., Department of Agriculture, Washington, D.C., personal communication (Nov. 1968).

51. Smith, W. S., and C. W. Gruber, Atmospheric Emissions from Coal Combustion—An Inventory Guide, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Division of Air Pollution (April 1966).

52. Snegireff, L. S., and O. M. Lombard, Arsenic and Cancer, Arch. Industr. Hyg. Occupational Med. 4:199 (1951).

53. Stanko, J. J., J. S. Wiseman, T. B. Wimberly, and O. Paganini, Some Air Pollution Problems Associated with Agriculture in Texas, J. Air Pollution Control Assoc. 18(3):164 (1968).

54. Stern, A. C., Air Pollution—Sources of Air Pollution and Their Control, vol. III, 2nd ed. (New York: Academic Press, p. 6831, 1968).

55. A Study of Air Pollution in Montana July 1961-July 1962, Montana State Board of Health, Division of Disease Control (1962).

56. Suggested Guide for the Use of Insecticides to Control Insects Affecting Crops, Livestock, Households, Stored Products, Forests and Forest Products - 1968, Agriculture Handbook No. 331, U.S. Dept. of Agriculture, U.S. Govt. Printing Office, Washington, D.C. (1968).

57. Swain, R. E., and W. D. Harkins, Arsenic in Vegetation Exposed to Smelter Smoke, J. Am. Chem. Soc. 30:915 (1908).

58. Thompson, R. J., G. B. Morgan, and L. J. Purdue, Analyses of Selected Elements in Atmospheric Particulate Matter by Atomic Absorption, Preprint, presented at the Instrument Society of America Symposium, New Orleans, La. (May 5-7, 1969).

59. Threshold Limit Values for 1967, Adopted at the 29th Annual Meeting of the American Conference of Governmental Industrial Hygienists, Chicago, Ill. (May 1-2, 1967).

60. USDA Summary of Registered Agricultural Pesticide Chemical Uses, Vol. 1, Herbicides, Defoliants, Desiccants and Plant Regulators, 3rd ed., Pesticides Regulation Division, Agricultural Research Service, U.S. Dept. of Agriculture, U.S. Govt. Printing Office, Washington, D.C. (1968).

OTHER REFERENCES

Air Quality Criteria for Particulate Matter, U.S. Dept. of Health, Education, and Welfare, National Air Pollution Control Administration Publication No. AP-49, U.S. Govt. Printing Office, Washington, D.C. (Jan. 1969).

Alarie, Y., "Screening Method for the Determination of Irritating Properties of Airborne Materials to the Upper Respiratory Tract," in Plethysmographic Method in Mice, Hazleton Labs, Inc., Falls Church, Va., Inhalation Division 1966).

Arnest, R. T., Atmosphere Control in Closed Space Environment (Submarine), Naval Medical Research Lab., New London, Conn., Bureau of Medicine and Surgery, Rept. No. 367 (Dec. 14, 1961).

Arnold, W., and P. Schroder, Losses in the Determination of Very Small Amounts of Arsenic and Their Radiochemical Evaluation, Text in German. Deut. Z. Ges. Gerichtl. Med. (Berlin) 59(2/3):305 (1967).

Arsine (Methods for the Detection of Toxic Substances in Air), Ministry of Labour, London Booklet No. 9 (1966).

Bamberger, E. S., and M. Gibbs, Effects of Phosphorylated Compounds and Inhibitors on $CO_2$ Fixation by Intact Spinach Chloroplasts, Plant Physiol. 40(5):919 (1965).

Bonser, G. M., Cancer Hazards of the Pharmacy, Brit. Med. J. 4:129 (1967).

Buechley, R. W., Epidemiological Consequences of an Arsenic Lung Cancer Theory, Am. J. Public Health 58(8):1228 (1963).

Chambers, L. A., M. J. Foter, and J. Cholak, A Comparison of Particulate Loadings in the Atmospheres of Certain American Cities, Proc. Natl. Air Pollution Symp., 3rd, Pasadena, Calif., pp.24-32 (1955).

Cuffe, S. T., and J. C. Knudson, Considerations for Determining Acceptable Ambient and Source Concentrations for Particulates from Cotton Gins, Preprint. Public Health Service, Cincinnati, Ohio (1965).

Heiman, H., Status of Air Pollution Health Research, 1966, Arch. Environ. Health 14:488 (1967).

Holland, R. H., and A. R. Acevedo, Current Status of Arsenic in American Cigarettes, Cancer 19(9): (1966).

Industrial Air Pollution Control, Heating, Piping, Air Conditioning 39(3):179 (1967).

Industry and Atmospheric Pollution in Great Britain, (Industrie et pollution atmospherique en Grande Bretagne) Centre Interprofessionnel Technique d'Etudes de la Pollution Atmospherique, Paris, France, Report No. CI 310, C.I.T.E.P.A. Document No. 24 (1967).

Ives, N. F., and L. Giuffrida, Investigation of Thermionic Detector Response for the Gas Chromatography of P, N, As, and Cl Organic Compounds, J. Assoc. Offic. Anal. Chemists 50(1)1 (1967).

Kambara, T., Y. Nohara, K. Ikegami, T. Nakamura, and T. Kajiwara, Arsine Poisoning: Report of an Autopsy Case, J. Sci. Labour (Tokyo) 42(6):454 (1966).

Katz, M., Recent Developments in Ambient Air Quality Guides in Relation to Control of Atmospheric Effluents, Pulp Paper Mag. Can. (Gardenvale) 69(4)60 (1968).

Lew, M., R. Woodruff, W. Johnson, and W. Musa, Ion Exchangers in Removal of Air Contaminants, San Francisco Bay Naval Shipyard, Vallejo, Calif., Chemical Lab. 8024-66, PR-1 (March 1967).

Lindberg, Z. Y., Arsenic Content on the Environment of a Superphosphate Plant, Hyg. Sanitation 29(7):119 (1964).

Lloyd, D. H., A Note on Factory Process Smells and Toxic Hazards, Sheet Metal Ind. (London) 44(481):331 (1967).

Pakhotina, N. S., Sanitary-Hygienic Evaluation of Industrial Emissions by a Zinc-Lead Combine, Survey of U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3: 93 (1960).

Posin, M. E., B. A. Kopylev, and N. A. Pebrova, Hydrogen Sulfide Absorption by Sodium Arsenate Solution in a Foam Apparatus, Zh. Prckl Kakim 31(6):849 (1958).

Sawicki, E., Airborne Carcinogens and Allied Compounds, Preprint, presented at the American Medical Association Air Pollution Medical Research Conference, Los Angeles, Calif. (March 2-4, 1966).

Stocks, P., On the Relations Between Atmospheric Pollution in Urban and Rural Localities and Mortality from Cancer, Bronchitis and Pneumonia, with Particular Reference to 3,4 Benzopyrene, Beryllium, Molybdenum, Vanadium and Arsenic, Brit. J. Cancer 14:397 (1960).

Stokinger, H. E., Effect of Air Pollutants on Wildlife, Conn. Med. 27(8):487 (1963).

Truhaut, R., Danger Thresholds, European Conference on Air Pollution, June 24-July 1, 1964, Council of Europe, Strasbourg, (1964).

Viniegra, G., Air Pollution, Text in Spanish. Salud Publica Mexico (Mexico) 8(4):601 (1966).

Wagoner, J. K., R. W. Miller, F. E. Lundin, Jr., J. F. Fraumeni, Jr., and M. E. Haij, Unusual Cancer Mortality Among a Group of Underground Metal Miners, New Engl. J. Med. 269: 284 (1963).

Weisburg, M. I., Air Pollution Control Field Operations Manual (A Guide for Inspection and Enforcement), Public Health Service, Division of Air Pollution, Washington, D.C. (1962).

# APPENDIX

APPENDIX

TABLE 6

PRODUCTION OF SELECTED PESTICIDES, UNITED STATES
(In Thousands of Pounds)

| Item | 1939 | 1945 | 1950 | 1955 | 1960 | 1965 | 1966 | 1967 |
|---|---|---|---|---|---|---|---|---|
| Calcium arsenate | 41,349 | 25,644 | 45,348 | 3,770 | 6,590 | 4,192 | 2,890 | 2,500 |
| Lead arsenate | 59,569 | 70,522 | 39,434 | 14,776 | 10,062 | 7,098 | 7,328 | 6,000 |
| White arsenic | 44,686 | 48,698 | 25,546 | c | c | c | c | c |
| Copper sulfate | 134,032 | 251,000 | 174,600 | 156,176 | 116,000 | 47,272 | 41,504 | 33,992 |
| Aldrin-toxaphene group[a] | | | | 77,025 | 90,671 | 118,832 | 130,470 | 120,183 |
| Benzene hexachloride[b] | c | c | 76,698 | 56,051 | 37,444 | c | c | c |
| DDT | c | 33,243 | 78,150 | 129,693 | 164,180 | 140,785 | 141,349 | 103,411 |
| Methyl bromide | | | | 9,222 | 12,659 | 14,303 | 16,345 | 19,665 |
| Methyl parathion | | | | | 11,794 | 29,111 | 35,862 | 33,344 |
| Parathion | | | c | 5,168 | 7,434 | 16,607 | 19,444 | 11,361 |
| Nabam | | | | | 2,978 | 2,489 | 2,053 | 1,361 |
| 2,4-D acid | c | 917 | 14,156 | 34,516 | 36,185 | 63,320 | 68,182 | 77,139 |

[a]Includes the chlorinated compounds, aldrin, dieldrin, endrin, chlordane, heptachlor, and toxaphene.

[b]Production of gamma isomer content in BHC was 17.1 million pounds in 1951, 10.7 million in 1955, and 6.9 million in 1960. Data in the table are on a gross basis.

[c]Not available.

APPENDIX

TABLE 7



ARSENICAL PESTICIDES RECOMMENDED FOR USE BY THE DEPARTMENT OF AGRICULTURE, 1968[a]

| Crop | Insecticides<br>Insect | Insecticide | Dosage[b] |
|---|---|---|---|
| Currant and Gooseberry | Imported currantworm | Lead arsenate | 3-4 |
| Strawberry | Slugs and snails | Calcium arsenate | 0.25-0.5 |
| Apple | Apple-and-thorn skeletonizer | Lead arsenate | 20 |
| | Apple maggot | Lead arsenate | 30 |
| | Bagworm | Lead arsenate | 30 |
| | Borer-roundheaded apple tree | Lead arsenate | 30 |
| | Cankerworms | Lead arsenate | 24 |
| | Codling moth | Lead arsenate | 30 |
| | Fall webworm | lead arsenate | 20 |
| | Fruitworms | Lead arsenate | 30 |
| | Leaf roller, red-banded | Lead arsenate | 30 |
| | Plum curculio | Lead arsenate | 30 |
| | Tent caterpillars, Eastern | Lead arsenate | 30 |
| Grape | Red-banded leaf roller | Lead arsenate | 9 |
| Pear | Borer, roundheaded apple tree | Lead arsenate | 30 |
| | Fruit-tree leaf roller | Lead arsenate | 32 |
| | Fruitworms | Lead arsenate | 30 |
| | Plum curculio | Lead arsenate | 30 |
| Plum and Prune | Eye-spotted bud moth | Lead arsenate | 24 |
| Asparagus | Cutworms | Calcium arsenate | 40 |
| Nonagricultural lands | Mosquito larvae | Paris green | 0.6-0.15 |

| Crop | Herbicides<br>Purpose | Herbicide | Dosage |
|---|---|---|---|
| Cotton | Preharvest dessicant | Arsenic acid | 4.4 |

[a]Information taken from Suggested Guide for the Use of Insecticides to Control Insects Affecting Crops, Livestock, Households, Stored Products, Forests and Forest Products—1968, Agriculture Handbook No. 331, U.S. Department of Agriculture, U.S. Govt. Printing Office (1968).

[b]Pounds of active ingredient to apply per acre.

APPENDIX

TABLE 8

QUANTITIES OF ARSENICAL PESTICIDES USED BY FARMERS IN 48 STATES, 1964

| Herbicides | Crops[a] (Thousands of pounds) | Other[b] (Thousands of pounds) | Total |
|---|---|---|---|
| Sodium, calcium, and zinc arsenites | 1,183 | 56 | 1,239 |
| Organic arsenicals | 1,006 | 71 | 1,007 |
| **Insecticides** | | | |
| Lead, calcium, maganesium, and manganese arsenates | 7,014 | 142 | 7,156 |
| **Defoliants and Desiccants** | | | |
| Arsenic acid | 4,973 | 136 | 5,109 |

[a] Includes all crops, pasture, and land in summer fallow.

[b] Includes fence rows, ditch banks, and other usages.

TABLE 9

ARSENIC AND LEAD CONCENTRATION IN THE AIR OF ORCHARDS[a] WHERE LEAD ARSENATE WAS USED AS AN INSECTICIDE

| Insecticide Operation | Concentration,[b] µg/m³ | | | |
|---|---|---|---|---|
| | Arsenic | | Lead | |
| | Average | Range | Average | Range |
| Mixing insecticide | 1,850 | 20-11,070 | 5,740 | 90-46,730 |
| Burning containers | 16,670 | 4,860-26,120 | 3,580 | 1,020-7,650 |
| Spraying orchard | 140 | 40-480 | 450 | 130-1,430 |
| Thinning fruit | 80 | 10-320 | 300 | 40-1,700 |
| Picking fruit | 880 | 260-1,900 | 2,930 | 770-7,520 |
| Dumping fruit | | | | |
| October | 60 | 10-190 | 190 | 40-690 |
| December | 10 | 2-20 | 30 | 1-110 |
| Sorting and packing (October) | 6 | 3-8 | 16 | 7-22 |

[a] Wenatchee, Wash. apple orchards (1938).

[b] Concentration to which orchardist was exposed.

APPENDIX

TABLE 10

SUSPENDED PARTICULATE AND ARSENIC CONCENTRATIONS IN THE AIR NEAR COTTON GINS IN WEST TEXAS, 1964[16]

| Distance from Gin (ft) | Range of Suspended Particulate Concentrations ($\mu g/m^3$) | Range of Arsenic Concentrations ($\mu g/m^3$) | Arsenic per µg Particulates Ratio X $10^4$ |
|---|---|---|---|
| 150-300[a] | 5,000-76,000 | 0.6-141 | 1.2-18.5 |
| 1,200-1,400[a] | 385-187 | .07-0.08 | 3.7-2.1 |
| 2,200-8,000[a] | 217-42 | .10-0.01 | 4.6-2.4 |
| [b] | 67-783 | | |
| | | | Ave. ~0.0003 |

[a]Measurement downwind from the gin.

[b]Measurement upwind from the gin.

TABLE 11

PARTICULATE EMISSIONS FROM STONEVILLE COTTON GIN[16]
(In Micrograms per Cubic Meter)

| Sampling Point | Settling Chamber | Sampling Filter | Total |
|---|---|---|---|
| Unloading fan | | 820,000 | 820,000 |
| Six-cylinder cleaner | 183,000 | 91,000 | 274,000 |
| Stick and bur machine | 1,190,000 | 68,000 | 1,258,000 |
| Seven-cylinder cleaner[a] | | 23,000 | 23,000 |
| Seven-cylinder cleaner[b] | | 11,000 | 11,000 |
| Condenser | | 46,000 | 46,000 |

[a]Standard cyclone, 84-inch diameter.

[b]High-efficiency cyclone, 34-inch diameter.

TABLE 12

ESTIMATED RATES OF EMISSION OF ARSENIC FROM COTTON GINS[16]

| Emission Rate of Arsenic | Minimum* | Maximum* |
|---|---|---|
| µg/min | 4,200-6,400 | 560,000-850,000 |
| µg/bale | 27,000-48,000 | 3,700,000-6,360,000 |

*Assuming efficiencies based on Stoneville Gin.

TABLE 13

AIR CONCENTRATION OF ARSENIC, 1950[8]

| City | Arsenic ($\mu g/m^3$) |
|---|---|
| Cincinnati | 0.06 |
| Charleston | <0.10 |

APPENDIX

TABLE 14

AIR CONCENTRATION OF ARSENIC IN CITIES OF UNITED STATES, 1953

| City | Population | Average Arsenic Concentration ($\mu g/m^3$) |
|---|---|---|
| Los Angeles | Over 2,000,000 | 0.02 |
| Detroit | " " | .04 |
| Philadelphia | " " | .16 |
| Chicago | " " | .04 |
| New York | " " | .05 |
| Cincinnati | 500,000-2,000,000 | .02 |
| Kansas City | " " | .02 |
| Portland | " " | .02 |
| Atlanta | " " | <.01 |
| Houston | " " | .01 |
| San Francisco | " " | .01 |
| Minneapolis | " " | .01 |
| Anchorage | <500,000 | <.01 |
| Charleston | " | .09 |
| Fort Worth | " | .01 |
| Louisville | " | .02 |
| Near Boonsboro | Nonurban area | .01 |
| " Salt Lake City | " | .03 |
| " Atlanta | " | .01 |
| " Cincinnati | " | <.01 |
| " Portland | " | .04 |
| Maximum urban concentration | | 1.41 |

APPENDIX

TABLE 15

CONCENTRATION OF ARSENIC IN AIR, FULL-YEAR DATA[2]

| Location | Year | No. of Samples | Micrograms per Cubic Meter | | | | | | | | | | | | Std Geom Dev |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Min | Frequency Distribution (Percent) 10 | 20 | 30 | 40 | 50 | 60 | 70 | 80 | 90 | Max | Arith Mean | Geom Mean | |
| Arizona | | | | | | | | | | | | | | | | |
| Phoenix | 1961 | 27 | | | | .01 | .01 | .01 | .01 | .02 | .03 | .04 | .11 | .02 | .01 | 2.63 |
| Delaware | | | | | | | | | | | | | | | | |
| Wilmington | 1961 | 23 | | | | .01 | .01 | .01 | .01 | .01 | .02 | .02 | .03 | .01 | .01 | 1.69 |
| District of Columbia | | | | | | | | | | | | | | | | |
| Washington | 1961 | 23 | | | | .01 | .01 | .01 | .01 | .02 | .02 | .03 | .04 | .02 | .01 | 1.79 |
| Georgia | | | | | | | | | | | | | | | | |
| Atlanta | 1962 | 25 | | | | | | .01 | .01 | .01 | .01 | .02 | .04 | .01 | .01 | 1.81 |
| Illinois | | | | | | | | | | | | | | | | |
| Chicago | 1962 | 26 | | | | .01 | .01 | .01 | .02 | .02 | .03 | .03 | .04 | .02 | .02 | 1.91 |
| Indiana | | | | | | | | | | | | | | | | |
| Gary | 1962 | 21 | | | .01 | .01 | .02 | .02 | .03 | .04 | .04 | .06 | .08 | .03 | .02 | 2.29 |
| Parke County | 1961 | 23 | | | | | | .01 | .01 | .01 | .01 | .01 | .02 | | .01 | 1.59 |
| New York | | | | | | | | | | | | | | | | |
| New York | 1961 | 23 | .01 | .01 | .01 | .02 | .02 | .02 | .03 | .03 | .04 | .05 | .11 | .03 | .03 | 1.85 |
| Pennsylvania | | | | | | | | | | | | | | | | |
| Clarion County | 1961 | 22 | | | | | .01 | .01 | .01 | .01 | .01 | .02 | .03 | .01 | .01 | 1.56 |
| Pittsburgh | 1961 | 23 | .01 | | .01 | .01 | .01 | .02 | .02 | .03 | .03 | .05 | .09 | .03 | .02 | 1.87 |
| Utah | | | | | | | | | | | | | | | | |
| Salt Lake City | 1961 | 18 | | | | .01 | .01 | .01 | .01 | .02 | .02 | .02 | .11 | .02 | .01 | 2.3 |

(continued)

APPENDIX

TABLE 15

CONCENTRATION OF ARSENIC IN AIR, FULL-YEAR DATA[2] (Continued)

| Location | Year | No. of Samples | Micrograms per Cubic Meter | | | | | | | | | | | | Std Geom Dev |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Min | Frequency Distribution (Percent) 10 | 20 | 30 | 40 | 50 | 60 | 70 | 80 | 90 | Max | Arith Mean | Geom Mean | |
| West Virginia Charleston | 1962 | 25 | | .01 | .03 | .05 | .08 | .20 | .22 | .36 | .38 | .53 | 1.0 | .24 | .11 | 4.84 |
| United States Urban | | 12* | <.01 | | | | | | | | | | 1.0 | | 0.02 | |

*Number of stations.

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3]

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| Alabama | | | | | |
| Birmingham | .02 | .01 | .00 | .02 | .01 |
| Gadsden | .01 | .01 | .00 | .01 | .01 |
| Mobile | .01 | .00 | .01 | .01 | .01 |
| Alaska | | | | | |
| Anchorage | .00 | .00 | .00 | .00 | .00 |
| Arizona | | | | | |
| Grand Canyon[a] | .00 | .00 | .00 | .01 | .00 |
| Paradise Valley | .01 | .00 | .00 | .02 | .01 |
| Phoenix | .01 | .00 | .00 | .02 | .01 |
| Tucson | .01 | .01 | .00 | .01 | .01 |
| Arkansas | | | | | |
| Little Rock | .00 | .00 | .00 | .00 | .00 |
| Montgomery County[a] | | .00 | .00 | .00 | |
| Texarkana | .00 | .00 | .00 | .01 | .00 |
| California | | | | | |
| Bakersfield | .01 | .00 | .00 | .01 | .01 |
| Burbank | .01 | .01 | .02 | .02 | .02 |
| Humboldt County[a] | .00 | .00 | .00 | .00 | .00 |
| Los Angeles | .01 | .00 | .01 | .01 | .01 |
| Monterey | .00 | .00 | .00 | .00 | .00 |
| Oakland | .01 | .00 | .01 | .02 | .01 |
| Calif. (continued) | | | | | |
| Pasadena | .01 | .01 | .02 | .02 | .02 |
| Sacramento | .00 | .00 | .01 | .00 | .00 |
| San Diego | .00 | .00 | .00 | .00 | .00 |
| San Francisco | .00 | .00 | .00 | .01 | .00 |
| Santa Ana | .01 | .00 | .01 | .01 | .01 |
| Santa Barbara | .00 | .00 | .00 | .01 | .00 |
| Colorado | | | | | |
| Denver | .01 | .00 | .01 | .01 | .01 |
| Montezuma County[a] | .00 | .00 | .00 | .00 | .00 |
| Connecticut | | | | | |
| Hartford | .01 | .01 | .01 | .02 | .01 |
| New Haven | .01 | .01 | .01 | .01 | .01 |
| Delaware | | | | | |
| Kent County | .01 | .01 | .01 | .01 | .01 |
| Newark | | | .01 | .01 | |
| Wilmington | .02 | .02 | .01 | .05 | .03 |
| District of Columbia | | | | | |
| Washington | .01 | .01 | .01 | .02 | .01 |

(continued)

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| Florida | | | | | |
| Florida Keys[a] | .00 | .00 | | .00 | |
| Jacksonville | .00 | .00 | .00 | .00 | .00 |
| Orlando | .00 | .00 | | .00 | |
| Tampa | .00 | .01 | .01 | | |
| Georgia | | | | | |
| Atlanta | .01 | .01 | .01 | .01 | .01 |
| Hawaii | | | | | |
| Honolulu | .00 | .00 | .00 | .00 | .00 |
| Idaho | | | | | |
| Boise | .00 | .00 | .00 | .01 | .00 |
| Buite County[a] | .00 | .00 | .00 | .01 | .00 |
| Illinois | | | | | |
| Chicago | .04 | .02 | .03 | .02 | .03 |
| Cicero | .03 | .02 | .02 | .02 | .02 |
| East St. Louis[b] | .03 | .01 | .04 | .02 | .03 |
| Moline | .02 | .01 | .00 | .01 | .01 |
| Peoria | .02 | .01 | .01 | .03 | .02 |
| Rock Island | .01 | .01 | .01 | .01 | .01 |
| Springfield | | | .00 | .01 | |
| Indiana | | | | | |
| Beverly Shores | .01 | .00 | .01 | .01 | .01 |
| Dunes PCL Post | .01 | .01 | .01 | .02 | .01 |

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| Indiana (continued) | | | | | |
| Dunes State Park | .01 | .01 | .00 | .01 | .01 |
| East Chicago | .03 | .05 | .04 | .05 | .04 |
| Evansville | .02 | .01 | .01 | .01 | .01 |
| Fort Wayne | .03 | .01 | .01 | .01 | .02 |
| Gary | .04 | .02 | .03 | .02 | .03 |
| Hammond[b] | .03 | .03 | .02 | .02 | .03 |
| Indianapolis | .04 | .01 | .01 | .02 | .02 |
| Ogden Dunes | .01 | .00 | .01 | .02 | .01 |
| Portage | .01 | .01 | .01 | .01 | .01 |
| Parke County[a] | .01 | .00 | .01 | .01 | .01 |
| Lafayette | .01 | .00 | .01 | .01 | .01 |
| Iowa | | | | | |
| Davenport | | .01 | .01 | .01 | |
| Delaware County[a] | .00 | .00 | .00 | .00 | .00 |
| Des Moines | .01 | .01 | .00 | .01 | .01 |
| Dubuque | .01 | .01 | .01 | .02 | .01 |
| Kansas | | | | | |
| Kansas City | .01 | .00 | .00 | .01 | .01 |
| Wichita | .01 | .01 | .00 | .01 | .01 |
| Kentucky | | | | | |
| Ashland | .04 | .03 | .02 | .04 | .03 |
| Covington | .01 | .01 | .02 | .01 | .01 |
| Louisville | .01 | .01 | .01 | .02 | .01 |

(continued)

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC AIR, 1964[3] (Continued)

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| Louisiana | | | | | |
| Baton Rouge | .01 | .00 | .00 | .01 | .01 |
| Lake Charles | .00 | .00 | .00 | .00 | .00 |
| New Orleans | .00 | .00 | .00 | .00 | .00 |
| Maine | | | | | |
| Acadia National Park[a] | .00 | .00 | .00 | .00 | .00 |
| Portland | .01 | .00 | .00 | .01 | .00 |
| Maryland | | | | | |
| Baltimore | .02 | .01 | .02 | .03 | .02 |
| Calvert | .00 | .00 | .00 | .01 | .00 |
| Massachusetts | | | | | |
| Boston | .02 | .01 | .01 | .01 | .01 |
| Springfield | .02 | .01 | .01 | .02 | .02 |
| Michigan | | | | | |
| Detroit | .03 | .03 | .02 | .02 | .03 |
| Wyandotte | .03 | .03 | .03 | | |
| Minnesota | | | | | |
| Duluth | .01 | .02 | .00 | .01 | .01 |
| Minneapolis | .01 | .01 | .00 | .00 | .01 |
| Moorhead | .01 | .00 | .00 | .01 | .01 |
| St. Paul | .01 | .00 | .00 | .01 | .01 |
| Mississippi | | | | | |
| Jackson | .00 | .00 | .00 | | |
| Jackson County[a] | | .00 | .00 | .00 | |
| Missouri | | | | | |
| Kansas City | .01 | .00 | .00 | .01 | .01 |
| St. Louis | .01 | .01 | .03 | .01 | .02 |
| Shannon County[a] | .00 | .00 | .00 | .00 | .00 |
| Montana | | | | | |
| Glacier National Park[a] | .00 | | .00 | .00 | |
| Helena | .02 | .00 | .02 | .05 | .02 |
| Nebraska | | | | | |
| Omaha | .04 | .02 | .03 | .02 | .03 |
| Thomas County[a] | .00 | .00 | .00 | .00 | .00 |
| Nevada | | | | | |
| Las Vegas | .00 | .00 | .00 | .00 | .00 |
| White Pine County[a] | .00 | .00 | .00 | .00 | .00 |
| New Hampshire | | | | | |
| Concord | .01 | .00 | .00 | .00 | .00 |
| Coos County[a] | .00 | .00 | .00 | .00 | .00 |

(continued)

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| New Jersey | | | | | |
| Bridgeton | | | .00 | .02 | |
| Camden | .03 | .02 | .02 | .04 | .03 |
| Glassboro | .01 | .01 | .00 | .02 | .01 |
| Jutland | .01 | .01 | | .01 | |
| Marlton | | | .02 | .02 | |
| Newark | .02 | .01 | .02 | .02 | .02 |
| New Brunswick | .01 | .03 | .04 | | |
| Pemberton | | .01 | .01 | .02 | |
| Perth Amboy | | .10 | .03 | .10 | |
| Princeton | | .01 | .01 | .02 | |
| Trenton | .01 | | .01 | .02 | |
| New Mexico | | | | | |
| Albuquerque | .01 | .00 | .00 | .01 | .01 |
| Colfax County[a] | .00 | .00 | .00 | .00 | .00 |
| New York | | | | | |
| Cape Vincent[a] | .01 | .01 | .01 | .01 | .01 |
| New York | .03 | .02 | .03 | .05 | .03 |
| North Carolina | | | | | |
| Cape Hatteras[a] | .00 | .00 | .00 | .00 | .00 |
| Charlotte | .01 | .00 | .00 | .01 | .01 |
| Fayetteville | .00 | .00 | .00 | .01 | .00 |
| Winston-Salem | .02 | .00 | .00 | .02 | .01 |

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| North Dakota | | | | | |
| Bismarck | .00 | .00 | .00 | .01 | .00 |
| Fargo | .01 | .00 | .00 | .01 | .01 |
| Ward County[a] | .00 | .00 | .00 | .00 | .00 |
| Ohio | | | | | |
| Akron | .04 | .04 | .04 | .05 | .04 |
| Cincinnati | .03 | .02 | .02 | .03 | .03 |
| Cleveland | .04 | .02 | .02 | .04 | .03 |
| Columbus | .02 | .02 | .02 | .03 | .02 |
| Dayton | .02 | .04 | .01 | .04 | .03 |
| Lorain | .03 | .02 | .01 | .02 | .02 |
| Steubenville | .04 | .07 | .08 | .15 | .09 |
| Toledo | .06 | .18 | .03 | .10 | .09 |
| Youngstown | .07 | .05 | .02 | .04 | .05 |
| Oklahoma | | | | | |
| Cherokee County[a] | .00 | .00 | .00 | .00 | .00 |
| Oklahoma City | .01 | .00 | .00 | .01 | .01 |
| Tulsa | .01 | .00 | .00 | .01 | .01 |
| Oregon | | | | | |
| Curry County[a] | | .00 | .00 | .00 | |
| Portland | .00 | .02 | .01 | .01 | .01 |

(continued)

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| Pennsylvania | | | | | |
| Bethlehem[b] | .02 | .01 | .01 | .01 | .01 |
| Clarion County[a] | | .01 | .01 | .02 | |
| Eagleville | | | .01 | .02 | |
| Embreeville | | .01 | .00 | .02 | |
| Lancaster | .02 | .01 | .01 | .02 | .02 |
| Philadelphia | .04 | .02 | .13 | .05 | .06 |
| Pittsburgh | .03 | .03 | .03 | .06 | .04 |
| Puerto Rico | | | | | |
| Guayanilla | .00 | .00 | .00 | .00 | .00 |
| Ponce | .00 | .00 | .00 | .00 | .00 |
| San Juan | .00 | .00 | .00 | .00 | .00 |
| Rhode Island | | | | | |
| Providence | .01 | .01 | .01 | .01 | .01 |
| Washington County[a] | .01 | .00 | .00 | .01 | .01 |
| South Carolina | | | | | |
| Columbia | .01 | | .01 | .01 | |
| Richland County[a] | .00 | .00 | .00 | .00 | .00 |
| South Dakota | | | | | |
| Black Hills | .00 | .00 | .00 | .01 | .00 |
| Sioux Falls | .01 | .00 | .00 | .00 | .00 |

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| Tennessee | | | | | |
| Chattanooga | .04 | .02 | .01 | .02 | .02 |
| Memphis | .02 | .01 | .01 | .01 | .01 |
| Nashville | .01 | .01 | .01 | .01 | .01 |
| Texas | | | | | |
| Arkansas County[a] | .00 | .00 | .00 | .00 | .00 |
| Dallas | .02 | .01 | .02 | .03 | .02 |
| El Paso | .50 | .60 | .50 | 1.40 | .75 |
| Houston | .01 | .00 | .01 | .01 | .01 |
| Laredo | | .01 | .00 | .01 | |
| San Antonio | .00 | .00 | .00 | .00 | .00 |
| Texarkana | .00 | .00 | .00 | .00 | .00 |
| Waco | .00 | .00 | .01 | .01 | .01 |
| Utah | | | | | |
| Ogden | .01 | .00 | .01 | .02 | .01 |
| Salt Lake City | .02 | .01 | .00 | .03 | .02 |
| Vermont | | | | | |
| Burlington | .01 | .01 | .01 | .01 | .01 |
| Orange County[a] | .00 | .00 | .00 | .01 | .00 |

(continued)

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

| Station Location | Micrograms per Cubic Meter | | | | |
|---|---|---|---|---|---|
| | 1st Qtr | 2nd Qtr | 3rd Qtr | 4th Qtr | Yrly Avg |
| Virginia | | | | | |
| Danville | .01 | .01 | .00 | .01 | .01 |
| Norfolk | .01 | .01 | .01 | .01 | .01 |
| Shenandoah Park[a] | .00 | .00 | .00 | .00 | .00 |
| Washington | | | | | |
| Seattle | .06 | .08 | .03 | .14 | .08 |
| Tacoma | .03 | .02 | .04 | .18 | .07 |
| West Virginia | | | | | |
| Charleston | .22 | .10 | .24 | .36 | .25 |
| Huntington | .03 | .02 | .01 | .03 | .02 |
| Parkersburg | .02 | .01 | .02 | .02 | .02 |
| Weirton | .01 | .03 | .05 | .05 | .04 |
| Wheeling[b] | .04 | .02 | .02 | .02 | .03 |
| Wisconsin | | | | | |
| Door County[a] | .00 | .00 | .00 | .01 | .00 |
| Eau Claire | .01 | .01 | .00 | .01 | .01 |
| Milwaukee | .02 | .02 | .01 | .02 | .02 |
| Racine | .01 | .01 | .01 | .02 | .01 |
| Superior | .01 | .01 | .00 | .01 | .01 |
| Wyoming | | | | | |
| Cheyenne | .00 | .00 | .00 | .00 | .00 |
| Yellowstone Park[a] | .01 | .00 | .00 | .02 | .01 |
| United States | 133 Stations | | | | .02 |

[a]Nonurban Areas

[b]1963

APPENDIX

TABLE 17

CONCENTRATION OF ARSENIC IN THE AIR OF MONTANA CITIES, 1961-62[55]

| | Arsenic, $\mu g/m^3$ (Average) | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| City | Maximum | July | Aug. | Sept. | Oct. | Nov. | Dec. | Jan. | Feb. | Mar. | Apr. | May | June |
| Anaconda | 2.50 | 0.42 | 0.68 | 0.51 | 0.33 | 0.54 | | 0.64 | 0.56 | 0.33 | 0.27 | 0.18 | 0.46 |
| Butte | 0.55 | .05 | .05 | .06 | .11 | .09 | 0.05 | .09 | .04 | .06 | .09 | .04 | .09 |
| Great Falls | 0.11 | .01 | .02 | .03 | .01 | .02 | .01 | .00 | .00 | .01 | .01 | .00 | .00 |
| Helena | 0.16 | .00 | .01 | .02 | .02 | .03 | .06 | .02 | .02 | .03 | .04 | .01 | .00 |
| Missoula | 0.15 | .00 | .00 | .01 | .00 | .02 | .01 | .02 | .01 | .02 | .00 | .00 | .00 |

APPENDIX

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS[33]

| Compound | Properties | Toxicity | Uses |
|---|---|---|---|
| Arsenic trioxide (White arsenic) $As_2O_3$ 75.74% | m.p. 313° b.p. 465° sublimes at 193° | Most forms of arsenic are highly toxic. Acute symptoms following ingestion relate to irritation of the gastrointestinal tract: nausea, vomiting, diarrhea which can progress to shock and death. Chronic poisoning can result in exfoliation and pigmentation of skin, herpes, polyneuritis, altered hematopoiesis, degeneration of liver and kidneys $LD_{50}$ for rats 13,000 µg/kg for man 0.0-0.5 g Chronic: Under 0.1 g Phytotoxicity: very toxic to plants. | It is the primary material for all arsenic compounds. Used in the manufacture of glass, Paris green, enamels, weed killers, textile mordants, metallic arsenic; for preserving hides, killing rodents, insects; in sheep dips and weed killers. Med. use: formerly for dermatides, chronic bronchitis, asthma, anemia, topically for skin neoplasms. Vet. use: for pulmonary emphysema, chronic coughs, anemia, general debility, chronic nonparasitic skin disease |
| Arsenic disulfide $As_2S_2$ 70.03% | m.p. 320° b.p. 565° | Most forms of arsenic are highly toxic See Arsenic trioxide | As pigment in painting, in fireworks as blue fire and to give an intense white flame; to manufacture shot; for calico printing and dyeing, tanning and depilating hides |
| Arsenic acid $H_3AsO_4$ 52.78% | | Poisonous. See above. $LD_{50}$ in rabbits: 8,000 µg/kg | In the manufacture of arsenates |

(continued)

APPENIDX

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

| Compound | Properties | Toxicity | Uses |
|---|---|---|---|
| Arsenic hemiselenide $As_2Se$ 65.49% | Decomposes | Same as Arsenic trioxide | In manufacture of glass |
| Arsenic pentafluoride $AsF_5$ 44.09% | m.p. $-79.8^{o}$ b.p. $-53.2^{o}$ | Same as Arsenic trioxide Also, external contact—liquid or vapor—causes severe irritation of eyes | |
| Arsenic pentaselenide $As_2Se_5$ 27.51% | Decomposes | Poisonous See Arsenic trioxide | |
| Arsenic pentoxide $As_2O_5$ 65.20% | | Poisonous. Keep well closed. LD i.v. in rabbits 8,000 µg/kg | In manufacture of colored glass, in adhesives for metals; in wood preservatives; in weed control; as fungicide |
| Arsenic tribromide AsBr 23.83% | m.p. $31.1^{o}$ | See Arsenic trioxide Intensely poisonous Also: highly irritating to skin, eyes, mucous membranes, respiratory tract | |
| Arsenic trichloride $AsCl_3$ 41.32% | m.p. $-16^{o}$ b.p. $130.21^{o}$ | Extremely toxic. Readily liberates hydrochloric acid, which is a strong irritant. Also see Arsenic trioxide | In the ceramic industry; in the synthesis of chlorine-containing arsenicals (i.e., chloro derivatives of arsenic) |

(continued)

APPENDIX

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

| Compound | Properties | Toxicity | Uses |
|---|---|---|---|
| Arsenic trifluoride<br>$AsF_3$<br>56.79% | m.p. -8.5°<br>b.p. 63° | Extremely toxic.<br>See Arsenic trioxide | |
| Arsenic triiodide<br>$AsI_3$<br>16.44% | m.p. 140.9°<br>b.p. ~400° | See Arsenic trioxide | Med. use: formerly internally for chronic dermatitides (including syphilitic), various chronic arthroses, and certain cases of lymphadenitis, and topically as stimulant in dermatitis |
| Arsenic triselenide<br>$As_2Se_3$<br>38.74% | m.p. 260° | See Arsenic pentafluoride | |
| Arsenic trisulfide<br>$As_2S_3$<br>60.90% | m.p. 300-325° | See Arsenic trioxide | In manufacture of glass, particularly infrared transmitting glass; in manufacture of oil cloth, linoleum; in electrical semiconductors, photoconductors; as pigment; for depilating hides, in pyrotechnics |
| Arsenious acid solution<br>Prepared with 1 g $As_2O_3$ 5 ml dil HCl and water to 100 ml | | See Arsenic trioxide | Med. use: has been used for blood dyscrasias.<br>Vet. use: in certain blood diseases, anemias, and skin disorders |

(continued)

APPENDIX

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

| Compound | Properties | Toxicity | Uses |
|---|---|---|---|
| Arsine<br>$AsH_3$<br><br>96.12% | m.p. -117°<br>b.p. -625° | Injurious in 1:20,000 dilution; a few inhalations may be fatal. Death from anoxia or pulmonary edema. Hemolytic anemia in non-fatal cases | For chemical analyses |
| Arsonoacetic acid<br>$H_2O_3AsCH_2COOH$<br><br>40.72% | m.p. 152° | | Vet. use: disodium salt used to treat anaplasmosis (babesiasis); as general stimulant in nervous disease; for eclampsia of bitches, and with adjuncts in chronic eczema and follicular mange |
| Calcium arsenate<br>$Ca_3(AsO_4)_2$<br><br>37.64% | Powder | See Arsenic trioxide<br>Poisonous<br>$LD_{50}$ 35-100 mg/kg for various animals<br>Phytotoxicity: causes leaf and fruit damage to stone fruit trees | As insecticide, particularly against insects destructive to plants; as molluscicide |
| Lead arsenate<br>$PbHAsO_4$<br><br>21.58% | White heavy powder<br>Decomposes above 280°C | Poisonous<br>$LD_{50}$ orally in rats 80,000 µg/kg<br>$LD_{50}$ animals 10,000-50,000 µg/kg<br>man 0.1-0.5 gm | As constituent of various insecticides for larvae of gypsy moth, boll weevil, etc.<br>Vet. use: has been reported useful for tapeworms of cattle, goats, sheep |

(continued)

APPENDIX

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

| Compound | Properties | Toxicity | Uses |
|---|---|---|---|
| Cupric acetoarsenite (Paris Green) $(CuOAs_2O_3)_3 \cdot Cu(C_2H_3O_2)_2$ 44.34% | Emerald green powder Decomposes on prolonged heating in water | Poisonous. Gastric disturbances, tremors, or muscular cramps, and peripheral neuritis, local effects on the skin, mucous membranes and conjunctivae | As insecticide, wood preservative; as pigment, particularly for ships and submarines |
| Cupric arsenite (Scheele's green) $CuHAsO_3$ 39.96% | Yellowish-gray powder | Poisonous | As pigment, wood preservative, insecticide, fungicide, rodenticide |
| Potassium arsenate $KH_2AsO_4$ 41.61% | Decomposes | Poisonous | In textile, tanning, and paper industries. In insecticidal formulations (especially fly paper) |
| Potassium arsenite $KAsO_2 \cdot HAsO_2$ 59.00% | Decomposes | Very poisonous. Keep well closed. $LD_{50}$ orally in rats: 14,000 µg/kg | In manufacture of mirrors to reduce the silver salt to metallic silver |
| Potassium arsenite solution Made from arsenic trioxide 10 g; potassium bicarbonate 7.6 g; alcohol 30 ml; distilled water to 1 liter | | Very poisonous See Arsenic trioxide | Med. use: has been used in chronic myelogenous leukemia, chronic dermatitides. Vet. use: for pulmonary emphysema, chronic coughs, anemia, general debility, chronic nonparasitic skin diseases |

(continued)

APPENDIX

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

| Compound | Properties | Toxicity | Uses |
|---|---|---|---|
| Methanearsenic acid $CH_3AsO(OH)_2$ 53.53% | m.p. 161° | See Arsenic trioxide | Disodium salt, as herbicide. Med. use: has been used in anemia, leukemia, psoriasis |
| Cacodylic acid $(CH_3)As(O)OH$ 54.29% | m.p. 195-196° | Poisonous LD s.c. for dogs 1.0 g/kg | As herbicide. Formerly for various skin diseases |
| Sodium arsenate dibasic $Na_2HAsO_4$ 40.29% | m.p. 57° | Poisonous but less so than arsenite | The technical grade, about 98% pure, is used in dyeing with Turkey-red oil and in printing fabrics. Med. use: formerly as "alterative," anthelmintic. Has been used for chronic skin diseases. Vet. use: see Arsenic trioxide |
| Sodium arsenite $NaAsO_2$ 57.67% | | Very poisonous. Keep well closed See Arsenic trioxide | The technical grade, 90-95% pure, is used in manufacture of arsenical soap for use on skin, for treating vines against certain scale disease, as insecticide (especially for termites). Vet. use: topically against ticks of ruminants |
| Chloroarsenol | m.p. 115° | | Formerly as tonic |

(continued)

APPENDIX

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

| Compound | Properties | Toxicity | Uses |
|---|---|---|---|
| Disodium methyl arsenate $CH_3AsO_3Na_2$- $6H_2O$ | m.p. above 300° | Oral to mammals: test animals tolerate well above 50,000 µg per kilo body weight $LD_{50}$ to rats 20% solution of agri. grade is 600,000 µg/kg | As weedkiller (crabgrass); for some control over silver or goose grass, knotweed, and chickweed |
| Zinc arsenate $Zn(AsO_4)_2$ 30% | | Oral to mammals See Arsenic trioxide | As insecticide |